CASE REPORT

Severe Exercise-Associated Hyponatremia on the Kokoda Trail, Papua New Guinea

Sean P. Rothwell, MBBS, FACEM; David J. Rosengren, MBBS, FACEM

From the Royal Brisbane and Women's Hospital, Queensland, Australia.

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Exercise-associated hyponatremia is the most common medical complication of ultradistance exercise and is usually caused by excessive hypotonic fluid intake. We report a case of severe hyponatremia in a healthy male trekking the Kokoda Trail in the remote Southern Highlands of Papua New Guinea. A 43-year-old male collapsed and had a generalized seizure in the afternoon of the third day of a guided trek. He was evacuated the following morning and was found to have a serum sodium level of 107 mmol/L on arrival to hospital. The case highlights that a high index of suspicion is required to identify patients with exercise-associated hyponatremia. Early diagnosis and appropriate management is critical to avoid the potentially fatal consequences of severe hyponatremia. The diagnosis and treatment of exercise-associated hyponatremia is particularly challenging in the remote Papua New Guinea jungle. Education of trek leaders, medics, and trekkers in appropriate preventative measures and the rapid treatment of exercise-associated hyponatremia is essential to avoid recurrences of this life-threatening condition.

Key words: hyponatremia, exertion, water intoxication, fluid therapy

Introduction

Exercise-associated hyponatremia (EAH) is a life-threatening metabolic imbalance. This condition has been previously described in competitors in endurance events, such as marathons and triathlons. We report a case of severe hyponatremia in a healthy male trekking the Kokoda Trail, Papua New Guinea.

The Kokoda Trail has gained increasing popularity for Australian travelers over the past decade. Traversing the Owen Stanley Range in the remote Southern Highlands of Papua New Guinea, the mountainous jungle trail was the site of a heroic rearguard action of Australian soldiers against the invading Japanese army in 1942. Most organized treks take between 6 and 9 days to complete the 96-km distance.

Corresponding author: Sean P. Rothwell, MBBS, FACEM, Emergency Physician Royal Brisbane and Women's Hospital, Butterfield Street, Herston, Queensland 4006 Australia (e-mail: sean.rothwell@health.qld.gov.au).

Case report

A 43-year-old male collapsed early in the afternoon on the third day of an 8-day guided trek over the Kokoda Trail in Papua New Guinea in August 2006. The patient complained of abdominal pains and leg cramps for 24 hours leading up to the collapse. He was noted to be weak and mildly ataxic during the trek on the third day. In the early afternoon, he collapsed suddenly and had a generalized seizure. Evacuation to a hospital was impossible until the following day owing to the remoteness of the Kokoda Trail. He remained semiconscious postictally, with an estimated best Glasgow Coma Score of 10 until the following morning.

The temperature at this time of year is between 19 and 25°C on average. Humidity is also high, with an average monthly rainfall of 165 mm. The first day of the trek is typically easy. The trek becomes physically difficult during the second day and remains challenging for the next 5 days. The patient's fluids for the trek consisted of plain water and water containing a sports drink powder. The exact volume of fluid ingested was not

known, but on the evening before and day of the collapse, fellow trekkers and guides encouraged him to drink large amounts of water.

He was physically fit prior to embarking on the Kokoda Trail, having completed several triathlons. His training regime consisted of climbing in mountainous terrain near Newcastle, New South Wales, while carrying a backpack. He had no previous medical conditions and was not on any regular medication. His malaria prophylaxis regime, which began 2 weeks before the trek, was doxycycline 100 mg twice daily.

He was evacuated by helicopter and taken to Port Moresby Hospital. On arrival, the patient's serum sodium was 107 mmol/L (135–145 mmol/L). Other investigation results included potassium 5.8 mmol/L (3.2–4.5 mmol/L), urea 12.8 mmol/L (3.0–8.0 mmol/L), and creatinine 108 μ mol/L (70–120 μ mol/L). He was treated with 3500 mL of 0.9% saline over approximately 36 hours. His condition improved, and he was transferred to a tertiary referral hospital in Australia. On arrival at this hospital, his sodium had increased to 132 mmol/L, and he was alert but still mildly confused.

He remained in the hospital a further 6 days. His creatine kinase peaked at 18 600 U/L (<200 U/L). Non-contrast-enhanced computed tomography of his head was normal. An electroencephalogram suggested a toxic or metabolic encephalopathy, while neuropsychological testing was consistent with mild hypoxic brain injury. He showed no clinical signs of central pontine myelinolysis. No magnetic resonance imaging was performed. His cognitive function improved during his admission. He returned to work 2 weeks after discharge.

Discussion

EAH is the most common medical complication of ultradistance exercise and is usually caused by excessive hypotonic fluid intake.^{1–13} There have also been several reports of EAH in moderate exercise events, such as day hiking and short triathlons.^{4,5} There are no documented cases of hyponatremia in trekkers on the Kokoda Trail.

The presenting signs and symptoms of hyponatremia from any cause include nausea, vomiting, headache, lethargy, muscle weakness, ataxia, confusion, pulmonary oedema, seizures, and death.^{1,5,9} Although caused by volume overload and volume depletion, respectively, EAH and dehydration share similar symptoms, including fatigue, headache, nausea, and vomiting. Both conditions are life-threatening in the wilderness. Incorrect diagnosis and treatment of one condition can potentially worsen the other. A history of adequate or large fluid intake, decreased thirst, moist mucous membranes, and altered

mental status should increase the clinical suspicion for EAH.^{1,5–7}

EAH is a modern phenomenon that has emerged with the increase in popularity of ultra-endurance events and encouragement to drink as much fluid as possible to avoid heat illness and improve performance.⁷ EAH is usually a dilutional hyponatremia, with an absolute increase in total body water. The primary aetiology is the consumption of hypotonic fluids in excess of body fluid losses.^{1–13} Other possible contributing factors include inappropriate antidiuretic hormone secretion during exercise and high sodium losses in sweat or urine.^{1,5,6,9,12}

Risk factors for the development of EAH are low body weight, female gender, greater than 4 hours of continuous exercise, slow performance pace, race inexperience, excessive drinking behavior, altered renal excretory capacity, and hot environmental conditions.^{1,6,8,12}

The initial treatment of symptomatic EAH is the avoidance of further isotonic or hypotonic fluids to prevent worsening of fluid overload. Those patients with more severe symptoms, such as confusion, seizures, or respiratory difficulty, should receive an initial bolus of 100 mL of hypertonic 3% NaCl solution. Although the use of hypertonic saline in the field has not been studied, the efficacy of this therapy has been well-documented in the hospital setting.^{1,8} The use of intravenous mannitol, if hypertonic saline is not available, has also been suggested.^{1,6} Despite these recommendations, this patient improved with isotonic saline treatment.

The patient's hyponatremia was likely exacerbated by fellow trekker's and guide's advice to continue hydration with water. Current recommendations for fluid intake in endurance events are to drink fluids only according to thirst (*ad libitum*).^{1,10,15} Universal guidelines with specific volumes have proven difficult to develop due to different environments, duration, and intensity of exercise and individual variation in sweat rates and renal water excretion.^{1,15} The consumption of electrolyte-containing fluids (sports drinks) has not been shown to prevent the development of EAH in athletes who drink to excess.^{11,16} There is disagreement in the literature regarding sodium supplementation. The Consensus Statement of the First International EAH Consensus Development Conference in 2005 states there is currently no evidence supporting the use of sodium supplementation to prevent the development of EAH.¹ The American College of Sports Medicine Position Stand on Exercise and Fluid Replacement 2007 recommends that sweat electrolyte losses should be fully replaced with beverages and snacks containing sodium to establish euhydration.¹⁵ Both groups agree that the avoidance of overdrinking will reduce the risk of EAH. Despite the large body of evidence to the contrary, this case demonstrates

the common fallacy even among experienced hikers and guides that large amounts of fluid are needed during prolonged exercise.

The wilderness is a common setting for endurance events, both competitive and noncompetitive. These include backcountry hiking, mountain biking, kayaking, and skiing, as well as combination multisport events. The intense physical challenge and the tangible history of the Kokoda Trail attracts up to 5000 trekkers of all ages each year, particularly from Australia. Walking the track typically consists of hiking up to 10 hours a day with a backpack weighing up to 20 kilograms for 7 to 10 days. The demographics of the Kokoda Trail trekkers differ from typical competitive endurance events. In one series of 211 trekkers, the average age was 45 years. A small number of them were taking medication known to cause hyponatremia, with 3 of them taking thiazide diuretics, 1 amitriptyline, 1 paroxetine, 1 fluoxetine, 1 carbamazepine, and 1 citalopram (Rosengren D, unpublished data, 2006). In addition, Kokoda trail trekkers have multiple known risk factors for developing EAH, including greater than 4 hours duration of exercise each day, slow performance pace, inexperience, hot environmental conditions, and excessive drinking behavior. Nonsteroidal anti-inflammatories (NSAIDs) are frequently used to treat joint and muscle pains caused by the arduous trek. Nonsteroidal anti-inflammatories have been linked to an increase in the incidence of EAH.¹⁷

This case highlights that a high index of suspicion is required to identify patients with EAH. Early diagnosis and appropriate management is critical to avoid the potentially fatal consequences of severe hyponatremia. The Kokoda Trail is fertile soil for the development of EAH, with its unique trekker demographic, geography, and climate. In addition, treating EAH is particularly challenging in the remote Papua New Guinea jungle. Education of trek leaders, medics, and trekkers in appropriate preventative measures and the rapid treatment of EAH is essential to avoid recurrences of this life-threatening condition.

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